# Surgical Treatment of Bleeding Esophageal Varices

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*• Massive bleeding from varices in the esophagus and stomach secondary to liver disease is a serious surgical emergency, as the patient may bleed to death. The problem is further complicated by the difficulties in making a diagnosis and the poor general condition of the patient due to the long standing liver disease. For the past two years at the San Francisco Hospital this problem has been handled by exploring the stomach and esophagus and ligating the bleeding point. No effort has been made to lower the pressure in the veins to the liver. The results have been sufficiently encouraging to warrant further trial.*

MASSIVE BLEEDING from the gastrointestinal tract secondary to liver disease is a serious problem that is faced relatively often in large city hospitals. The various forms of surgical treatment now in use to stop bleeding of this kind are: Ligation of splenic artery; ligation of hepatic and splenic arteries; injection of varices with sclerosing solutions; esophagogastrectomy and total gastrectomy; esophageal and gastroesophageal tamponade; and ligation of varices transthoracically. Varying degrees of success have been reported from each of the methods. It is the purpose of this presentation to report experiences in 12 cases in which an attempt was made to control the bleeding during the hemorrhage by transthoracic ligation of the bleeding point.

Direct surgical attack on bleeding varices has been described by Boerema (cited by Linton and Warren[7]), Crile[5] and Linton and Warren.[7]

The surgical problem is complicated by two important factors: the poor condition of the patients with consequent high fatality rate from the hemorrhage, and the uncertainty of the diagnosis in many instances. The diagnosis of esophageal varices is by no means certain unless one is fortunate enough to have had previous gastrointestinal or esophagoscopic examination of the patient. The presence of cirrhosis of the liver, while helpful, does not rule out an ulcer of the stomach or duodenum as a source of the hemorrhage. At autopsy of 68 patients with hepatic cirrhosis who died from hemorrhage at the San Francisco Hospital, bleeding was shown to have come from a duodenal ulcer in four patients. This well known association of cirrhosis and ulcer was most recently pointed out by Fainer and Halsted.[6] Twenty-nine per cent of the patients they reported upon had two clinically demonstrable lesions.

Esophageal varices cannot be demonstrated in all patients with cirrhosis who bleed. In the previously mentioned autopsy series, 23 of the 68 patients had no demonstrable cause of bleeding. Conversely, esophageal varices can occur in patients who do not have cirrhosis. Failure to demonstrate varices may be due to the fact, as Palmer and Brick[8] have shown, that autopsy and x-ray techniques are too crude. They were able to demonstrate varices by esophagoscopic means in 22 patients who did not have cirrhosis of the liver. It is interesting that in a few of their cases in which the portal pressure was measured through the esophagoscope, the measurements were within the normal pressure range.

In an attempt to solve this diagnostic problem, some clinicians have utilized the Sengstaken tube.[10] With the balloon in place, material aspirated from the stomach would show blood in patients with bleeding ulcer. The author's experience with the use of the Sengstaken tube is limited, but it has been found to be difficult to place and difficult to hold in place. Moreover, blood was aspirated in some cases even when the bleeding was proved to be from varices. One patient vomited up the balloon in the distended state, and in two patients bleeding began again the moment the pressure was released.

Although the author has not had much experience with gastroesophageal barium examination in the presence of massive hemorrhage, it is difficult to see how a roentgenologist can do an adequate examination on a patient bordering on shock whose stomach may be filled with clots.

Because of this difficulty in accurate diagnosis of the source of hemorrhage, it is felt that in every case in which operation for control of bleeding is done, the stomach ought to be opened. The most adequate approach to the stomach and lower esophagus, as well as the best exposure if a splenorenal anastomosis is necessary, is through a left thoracoabdominal incision.

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In the case of a few patients with atypical history in the present series, 75 cc. of 35 per cent Diodrast® was injected into the spleen. This is a simple procedure, and the x-ray film taken at that time may demonstrate an extrahepatic obstruction (as shown in Figure 2), the only indication for a decompression operation in addition to ligation of the bleeding point.

It is important from the surgical point of view that the patients with bleeding are likely to be in extremely poor state of nutrition and in many instances cannot live long if bleeding continues. This is true generally of patients with cirrhosis. The onset of the hemorrhage in many of these patients seems to occur after a long bout of alcoholism.

Table 1 gives data on the patients in the present series who were operated upon with a presumptive diagnosis of hemorrhage from esophageal varices. It is of interest that in three patients the bleeding point was from a gastric varix. In one of these patients the gastric varix was in a hiatus hernia. There were two cases of extrahepatic obstruction, one certain and one questionable. There were two deaths. One patient died during induction of anesthesia from vomiting and aspiration. Thereafter in all cases the intratracheal tube was placed under local anesthesia before the operation was started. The second death occurred on the twelfth postoperative day and was due to leakage of the gastrotomy suture line with mediastinitis in a severely cirrhotic patient who also had delirium tremens postoperatively.

A left thoracoabdominal incision is used for best exposure of the lower esophagus and stomach. At operation the spleen usually is removed first to facilitate exposure. The splenic vein is preserved for possible anastomosis. The pressure is measured

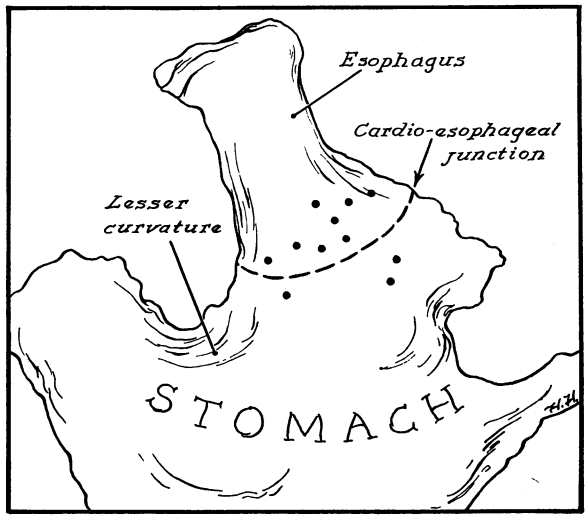


Figure 1.—Diagram showing source of hemorrhage in those cases where a single point was found to be bleeding.

directly in the portal system and the stomach is opened high, near the esophageal junction. If no evidence of ulcer is found in the stomach or duodenum, the esophageal hiatus is exposed with anal retractors. If hemorrhage is in progress, locating the source of bleeding is not difficult, but it may not be from a single point. If the bleeding varix can be discovered, it is sutured with a running stitch.

Curiously, although in many cases varices extend the entire length of the esophagus, the bleeding seems to occur invariably either in the stomach or esophagus, within about 5 cm. of the cardioesophageal junction. While it is possible that esophagitis is the cause of hemorrhage in cases in which the bleeding comes from an esophageal varix, it certainly does not explain the bleeding from gastric varices. Undoubtedly there are many causes for the

**TABLE 1.—*Data on 12 cases in which transthoracic operation was done to stop bleeding from esophageal varices***

| Patient | Age; Sex | Diagnosis | Bleeding After Suture | Deaths | Follow-up (Months) |
|---|---|---|---|---|---|
| 1 | 54 M | Cirrhosis | 0 | 0 | 11 |
| 2 | 61 M | Cirrhosis. Gastric varix. | 0 | 0 | 14 |
| 3 | 44 M | Cirrhosis | Yes | 0 | 6 |
| 4 | 9 F | Cirrhosis (PCA 1952) | 0 | Yes | Cholemia 6 |
| 5 | 28 M | Extrahepatic obstruction | No bleeding source demonstrable | 0 | 6 |
| 6 | 38 M | Cirrhosis. Gastric varix | 0 | 0 | 9 |
| 7 | 65 M | Cirrhosis | 0 | 0 | 14 |
| 8 | 64 M | Banti's syndrome ? Marginal ulcer ? Extrahepatic obstruction | Yes | 0 | 2 |
| 9 | 68 M | Cirrhosis | 0 | 0 | 18 |
| 10 | 62 M | Cirrhosis | 0 | 0 | 5 |
| 11 | 50 M | Cirrhosis | 0 | Yes | 12 days |
| 12 | 62 F | Cirrhosis | Yes | 0 | 11 |
| 13 | 47 F | Cirrhosis | No bleeding source demonstrable 0 | Yes | Died during induction of anesthesia |

hemorrhage in such cases. One patient was observed in whom hemorrhages occurred twice in small amounts after a ligation operation, and both times the bleeding followed a severe bout of coughing.

Figure 1 is a diagram illustrating the point of obstruction in the 11 cases in which a single source of bleeding was discovered.

Following are reports of illustrative cases.

CASE 1. *Extrahepatic obstruction, proven by x-ray; splenorenal anastomosis.*

An 18-year-old white man was sent to the hospital on January 20 because of gastrointestinal bleeding. The patient had been well until January 18, when he reported to the surgical clinic for evaluation of an inguinal hernia. At that time an indirect hernia was felt on the right side. The patient stated that he had had an enlarged spleen since the age of seven, but he did not want operation for the spleen since, he said, it had been taken care of by previous treatment. (The spleen could be felt on deep palpation.) The patient left the surgical clinic and that night felt weak and defecated black stools. The following day he again felt weak and had a black stool. On the morning of January 20 he became nauseated and vomited about four times. The first vomitus was dark brown in color and subsequent vomitus contained bright red blood. The hemoglobin content at that time was 7.5 gm. per 100 cc. of blood. Two days before it had been 10 gm.

The patient had had the first episode of hematemesis and melena at age 7, when he was kicked in the stomach by a playmate. A second episode occurred at age 8 when he did some heavy lifting. At age 12 and 15 he again had episodes of bleeding. The enlarged spleen first was noted on physical examination at age 12.

The patient was well developed and well nourished but pale. The spleen was palpable four finger-breadths below the costal margin. It was firm and slightly tender. The liver and other organs were not palpable. The blood pressure was 130/60 mm. of mercury and the pulse rate 100. An indirect inguinal hernia was present on the right side.

On January 20 and 21 the patient was given six blood transfusions of 500 cc. each and the hemoglobin content rose to 11.9 gm. per 100 cc. Esophagoscopy was done on the afternoon of January 20 and varices were seen, but no actual bleeding point was demonstrated. The patient vomited several hundred cubic centimeters of dark reddish-brown material during the procedure. A Sengstaken tube was inserted. On January 22, following the transfusions as noted above, the patient was doing quite well and the esophageal balloon was deflated and old blood returned from the stomach. The patient was given another 500 cc. of blood at that time. The esophageal balloon was reinflated. On January 24, with bleeding continuing, another 1,500 cc. of blood was required and, accordingly, surgical treatment was instituted. A preoperative hepatogram was made (Figure 2).

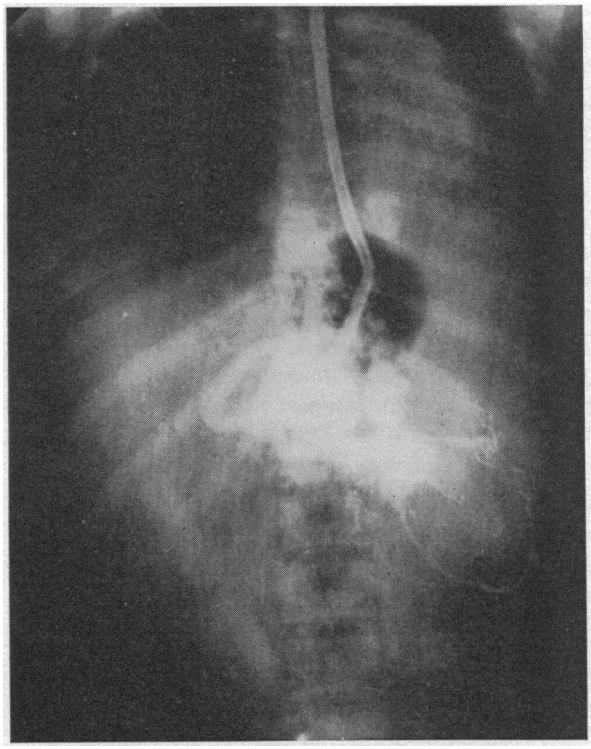

**Figure 2.—The hepatogram in Case 1, showing no dye in the liver. The spleen is outlined. Interpretation: Complete extrahepatic obstruction. Operation: Splenorenal anastomosis.**

The chest and abdomen were opened through a combined thoracoabdominal incision through the eighth interspace, the diaphragm was opened and the spleen was exposed. The spleen was approximately five times normal size. Splenectomy was carried out, the splenic vein being preserved. This vein was not thrombosed. The stomach was opened on the anterior surface and very large varices were observed. No attempt at ligation was made as no one bleeding point could be seen, and the stomach was closed. The pressure, measured in a tributary of the splenic vein, was 345 mm. of water. Splenorenal anastomosis was carried out. Following this procedure the pressure, determined in one of the tributaries of the splenic vein, was 210 mm. of water. The liver was normal in appearance and was not enlarged. The patient made an excellent postoperative recovery. The Levine tube was removed four days postoperatively. The hemoglobin content following operation was 13.1 gm. per 100 cc. and did not again decline. The sutures were removed on the seventh postoperative day and the wound was well-healed.

CASE 2. *Hemorrhages from a gastric varix within a hiatus hernia in a cirrhotic.*

A 50-year-old white man was admitted to the hospital on February 9. He had been vomiting blood every hour since awakening on the morning of entry. The week before entry the patient noted black stools several times daily.

The patient had been admitted to hospital thrice previously in the preceding four years for Laennec's cirrhosis, alcoholism, ascites, edema, avitaminosis and bleeding esophageal varices.

When examined the patient was sitting up in bed. Slight flaccidity of the face and gross tremor of the arms were noted. There were vascular spiders over the nose, left cheek and anterior chest and increased venous markings of the right anterior chest and right abdomen.

The patient continued to vomit blood and had tarry stools. He went into shock with blood pressure at 66/44 mm. of mercury on the evening of entry, and after two units of blood had been given he was taken to surgery. Through a left thoracoabdominal incision gastrotomy was performed and several actively bleeding varices were ligated with gut sutures in the region of the cardia of the stomach. There was believed to be a small hiatal hernia present also. No ulcers were noted. The spleen was removed. A specimen of the liver was taken for biopsy. The patient received six units of blood during operation.

Postoperatively, the patient had fever, subcutaneous emphysema and purulent exudate from the chest wound. Thoracentesis was carried out February 16 and 65 cc. of serosanguinous material was removed. It contained Gram-positive streptococci and staphylococci. Cultures were not reported. On February 18 dehiscence of the thoracic wound developed and a sucking sound could be heard. Left lower lobe pneumonia with three fluid levels and definite empyema were noted in an x-ray film of the chest. Penicillin, streptomycin and supportive intravenous therapy were given but the patient died on February 21.

At autopsy a leak was noted at the suture line in the gastrotomy wound and mediastinitis was present. The bleeding varix, which was thrombosed, was located in the gastric side within a small esophageal hiatal hernia. The liver was cirrhotic.

CASE 3. *Hemorrhage from a gastric varix. Mild cirrhosis.*

A 64-year-old male Negro prisoner entered the hospital in November 1953 because of massive hematemesis of a few hours' duration. At a previous entry a diagnosis of suspected cirrhosis of the liver had been made. No abnormalities were noted in a gastrointestinal series done at that time.

A presumptive diagnosis of bleeding ulcer was made and operation was done. Only slight pathologic changes were noted on biopsy of the liver. When the stomach was opened a small fountain of blood was seen pouring from one of several gastric varices. The varix was stitch ligatured.

After recovery the patient was sent back to prison, where he has had no further hemorrhage in 14 months.

CASE 4. *Repeated hemorrhages from varices. Cause undetermined.*

A 59-year-old white man entered the hospital in February 1955 for the ninth time with gastrointestinal bleeding. He had had this condition intermittently since 1947. In 1948 a gastric resection was done at another hospital, but no ulcer had been demonstrated. The patient first entered San Francisco Hospital in 1951. No blood dyscrasia had ever been demonstrated, although a diagnosis of erythroblastic anemia had been suggested. The spleen was large and esophageal varices had been demonstrated roentgenographically. On the eighth entry in September 1954 for severe hemorrhage, operation was performed. The liver did not appear grossly diseased. Portal pressures were within normal limits. The spleen was removed and the stomach opened. No marginal ulceration was found. There was some oozing from some small veins in the distal esophagus. These were stitched. In February 1955, after a drinking bout, the patient again vomited some blood, but on supportive therapy the hemorrhage stopped.

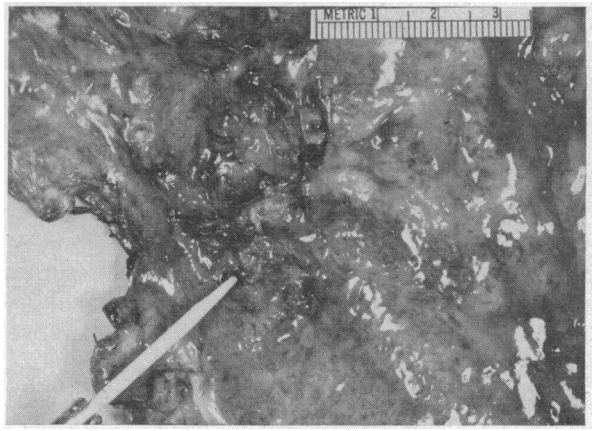


Figure 3.—The thrombosed varix in Case 2. The bleeding point was within a hiatal hernia. The sutured varix can be seen parallel to the gastroesophageal junction.

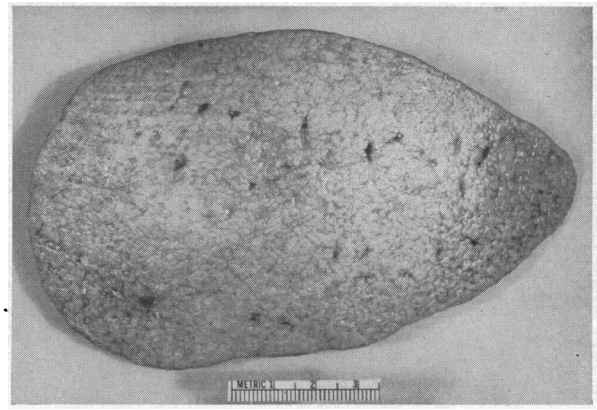

Figure 4.—The appearance of the liver in Case 2.

## DISCUSSION

The small series of cases reported would suggest that direct attack on bleeding esophageal varices is feasible. There does not seem to be any doubt about

this fact in the case of the patient with extrahepatic portal obstruction as the cause of esophageal varices. In these patients, splenorenal anastomosis should be done at the time of operation.

In the case of patients with cirrhosis of the liver, the problem is not quite so clear cut. It is obvious that ligation of one varix where several are present hardly can be expected to control the bleeding more than temporarily. Linton and Warren expressed the opinion that a portacaval anastomosis should be done after ligation, at some time in the postoperative period when the patient's condition has improved.

The author has not done portacaval anastomosis, for it is felt that lowering of the blood flow to the liver in the presence of cirrhosis is as damaging in these poor risk patients as any possible theoretical lowering of the pressure in the varices may be in the prevention of further hemorrhage. It is therefore of interest to note that the surviving patients in the series here reported, who have not had any form of portal decompression, seem to have about the same results as those reported by Linton and Warren who had portal decompression.

#### ADDENDUM

Since the presentation of this report, three of the surviving patients have bled again in small amounts. In one patient with large varices demonstrable by x-ray, portacaval anastomosis has been done.

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